

Effects of different heavy-resistance exercise protocols on plasma β -endorphin concentrations

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KRAEMER, WILLIAM J., JOSEPH E. DZIADOS, LOUIS J. MARCHITELLI, SCOTT E. GORDON, EVERETT A. HARMAN, ROBERT MELLO, STEVEN J. FLECK, PETER N. FRYKMAN, AND N. TRAVIS TRIPLETT. *Effects of different heavy-resistance exercise protocols on plasma β -endorphin concentrations*. J. Appl. Physiol. 74(1): 450-459, 1993.—To examine the changes of plasma β -endorphin (β -EP) concentrations in response to various heavy-resistance exercise protocols, eight healthy male subjects randomly performed each of six heavy-resistance exercise protocols, which consisted of identically ordered exercises carefully designed to control for the repetition maximum (RM) resistance (5 vs. 10 RM), rest period length (1 vs. 3 min), and total work (joules). Plasma β -EP, ammonia, whole blood lactate and serum cortisol, creatine kinase, urea, and creatinine were determined preexercise, midexercise, immediately postexercise, and at various time points after the exercise session (5 min-48 h), depending on the specific blood variable examined. Only the high total work-exercise protocol [1 min rest, 10 RM load (H10/1)] demonstrated significant increases in plasma β -EP and serum cortisol at midexercise and 0, 5, and 15 min postexercise. Increases in lactate were observed after all protocols, but the largest increases were observed after the H10/1 protocol. Within the H10/1 protocol, lactate concentrations were correlated ($r = 0.82$, $P < 0.05$) with plasma β -EP concentrations. Cortisol increases were significantly correlated ($r = 0.84$) with 24-h peak creatine kinase values. The primary finding of this investigation was that β -EP responds differently to various heavy-resistance exercise protocols. In heavy-resistance exercise, it appears that the duration of the force production and the length of the rest periods between sets are key exercise variables that influence increases in plasma β -EP and serum cortisol concentrations. Furthermore the H10/1 protocol's significant challenge to the acid-base status of the blood, due to marked increases in whole blood lactate, may be associated with mechanisms modulating peripheral blood concentrations of β -EP and cortisol.

anaerobic; opioid peptides; creatine kinase; lactate; cortisol; ammonia

High-intensity maximal exercise may present a physiological involvement of endorphins different from that caused by submaximal endurance exercise stress. Kjaer et al. (22) showed that the depressive effect of epidural blockade on β -EP concentrations was less pronounced with maximal than with submaximal exercise. It was suggested that with maximal exercise, aside from impulses from the muscles, nervous impulses from the motor centers in the brain are major determinants of neuroendocrine secretion and enhanced activity in higher neuroendocrine centers. Thus it appears that maximal short-term exercise presents a neurophysiological exercise stress that is much different from that caused by submaximal endurance exercise. Farrell et al. (13) suggested that when an organism is stressed, both the endorphin and the sympathetic nervous systems are activated, and one purpose of the endorphin activation may be to modulate the amount of sympathoactivation.

The intensity of the exercise has been demonstrated to be a primary variable involved in the stimulatory influence on plasma β -EP concentrations with exercise (3, 15, 19, 20). Still, because of the inverse relationship between exercise intensity and duration of exercise, it appeared that a certain duration of exercise would be needed to stimulate a significant increase in plasma concentrations of β -EP in a single bout of high-intensity exercise. Kraemer et al. (28) observed that when exercise intensity was increased from 115 to $\geq 175\%$ of maximal O_2 consumption ($VO_{2\max}$), significant increases in plasma β -EP were no longer observed. Heavy-resistance exercise provides an exercise model for one of the most common forms of high-intensity exercise, which is performed well above the level that produces $VO_{2\max}$ but uses multiple exercise bouts. Although the duration of exercise is quite short and the intensity very high, multiple bouts of heavy-resistance exercise using a large amount of total muscle mass usually make up a typical exercise session (23, 27) and may provide the necessary total duration of exercise needed to stimulate significant increases in plasma β -EP.

In a previous investigation, we demonstrated that the exercise stimulus used in heavy-resistance exercise can be carefully quantified and related to the configuration of the exercise variables (26). Furthermore it appeared

OVER THE PAST 15 years, endurance exercise has been the primary focus of research relating exercise stress to changes in the peripheral blood concentrations of β -endorphin (β -EP). It has become evident that higher intensities of endurance exercise stress produce increases in plasma concentrations of β -EP both during and after exercise (6, 15, 19, 20, 24, 25).



1098

that the hypothalamic-pituitary axis was very sensitive to changes in a heavy-resistance exercise protocol. Our previous observations demonstrated that dramatic increases in serum growth hormone resulted when the duration of the exercise sets (i.e., no. of repetitions) was increased [i.e., rather than using a weight that would allow only 5 repetitions, called the 5 repetition maximum (5 RM), the weight was lightened to allow only 10 repetitions (10 RM)] and rest periods between sets and exercises were reduced (from 3 to 1 min) (26). Only in an early study by Elliot et al. (12) were β -EP responses to heavy-resistance exercise examined. Still, the interpretation of significant increases in plasma β -EP/ β -lipotropin after heavy-resistance exercise remained unclear, inasmuch as data from this study were confounded by β -lipotropin cross-reactivity and the use of only one resistance exercise protocol. Thus it was unclear whether resistance exercise per se would provide the necessary stimulus for increased plasma concentrations of β -EP or whether the characteristics of the exercise protocol utilized were vital, as previously observed with growth hormone. It was hypothesized that increases in plasma β -EP concentrations would follow a pattern similar to growth hormone and be most sensitive to the same long-duration and short-rest protocol that was used previously and elicited the greatest response of the hypothalamic-pituitary axis. Therefore the primary purpose of the present study was to examine the changes in plasma concentrations of β -EP in response to the same quantified array of heavy-resistance exercise protocols used previously (26). This would help extend our understanding of the influence of exercise variables on peripheral plasma β -EP concentrations with this type of high-intensity intermittent resistance exercise stress. It might also provide the basis for further investigations into the physiological mechanisms involved with increases in plasma β -EP with high-intensity exercise.

METHODS

Within the context of our previous investigation (26), eight of the nine male subjects were utilized in this investigation to examine the plasma β -EP response patterns to the same combination of heavy-resistance exercise protocols used previously. Each subject gave informed written consent to participate in the investigation. The physical characteristics of the subjects were as follows (means \pm SE): age, 24.7 ± 1.6 yr; height, 178.4 ± 2.9 cm; body mass, 82.1 ± 4.4 kg; $\dot{V}O_{2\max}$, 55.2 ± 0.8 ml \cdot kg $^{-1}$ \cdot min $^{-1}$; and body fat, $16.1 \pm 1.6\%$. All subjects were in good health and had recreational experience with resistance training, but none were competitive lifters. The subjects had no history of any endocrine disorders or drug use and were not on any medications or nutritional supplementation during the course of the investigation.

Except for the dependent variables, the times when blood samples were obtained, and biochemical methodologies, the methods of the study were identical to those previously described (26). A minimum of 2 wk was taken for experimental protocol familiarization, descriptive testing, and load verification for each exercise protocol. Body composition was determined by hydrostatic weigh-

ing with use of a computer-interfaced load cell and standard body composition methodology (16, 37). $\dot{V}O_{2\max}$ (ml \cdot kg $^{-1}$ \cdot min $^{-1}$) was determined utilizing a continuous treadmill protocol (9, 36).

Experimental design and exercise protocols. Each of the six heavy-resistance exercise protocols was performed in random order and by all eight subjects. Subsequent statistical analysis demonstrated no order effects. The design allowed for more quantitative examination of the effects of specific program design variables (load and rest period length) corrected for total work. Figure 1 overviews the basic experimental design of the two series of exercise protocols used in the investigation. Two exercise series were used in this study, each consisting of three workouts (i.e., a primary workout, a rest control, and a load control). The strength (S) series was characterized by heavier resistance (i.e., 5 RM) in the primary workout. The hypertrophy (H) series used a lighter resistance (i.e., 10 RM) in the primary workout but had a higher volume of total work. The S series had significantly ($P < 0.05$) lower total work than the H series ($49,980 \pm 10,473$ vs. $60,427 \pm 13,428$ J).

As in our previous study (26), the primary workout in the S series consisted of a 5-RM load and a 3-min rest period between sets and exercises. It was designated S5/3, meaning S for the S series (lower total work), 5 for the 5-RM load, and 3 for the 3-min rest periods. The load control workout for the S series used a 10-RM load and was designated S10/3. The rest control workout for the S series used a 1-min rest period and was designated S5/1. The same type of terminology was again utilized to designate each exercise protocol in the H series. The primary protocol for the H series was designated H10/1, meaning H for H series (higher total work), 10 for the 10-RM load, and 1 for the 1-min rest periods between sets and exercises. Similarly, the load control workout used a 5-RM load and was designated H5/1, and the rest control workout used 3-min rest periods and was designated H10/3. Although both exercise protocols produce increases in strength and muscle cell hypertrophy, the S5/3 workout is typical of weight-training protocols used primarily for "strength" development and the H10/1 workout is typical of exercise regimens used by body builders to induce increases in muscular hypertrophy (27). Thus each exercise series provides for specific program differences between exercise protocols on the basis of the configuration of the exercise stress variables. The variations of the primary workouts were examined to help determine whether differences in responses occurred because of single factor changes in load and rest period lengths. Comparisons between a few S and H series protocols (S10/3 vs. H10/3 and S5/1 vs. H5/1) allowed a limited evaluation of total work effects. The exercises utilized, the order used, and the number of sets for the primary workouts can be seen in Table 1. Each of the two exercise series had a primary workout (S5/3 and H10/1), a load control (S10/3 and H5/1), and a rest control (S5/1 and H10/3). All workouts within a series involved the same total work.

The grip width used by each subject was proportional to his height. Body position of all subjects (e.g., grip width, joint angles) was held constant for an exercise in

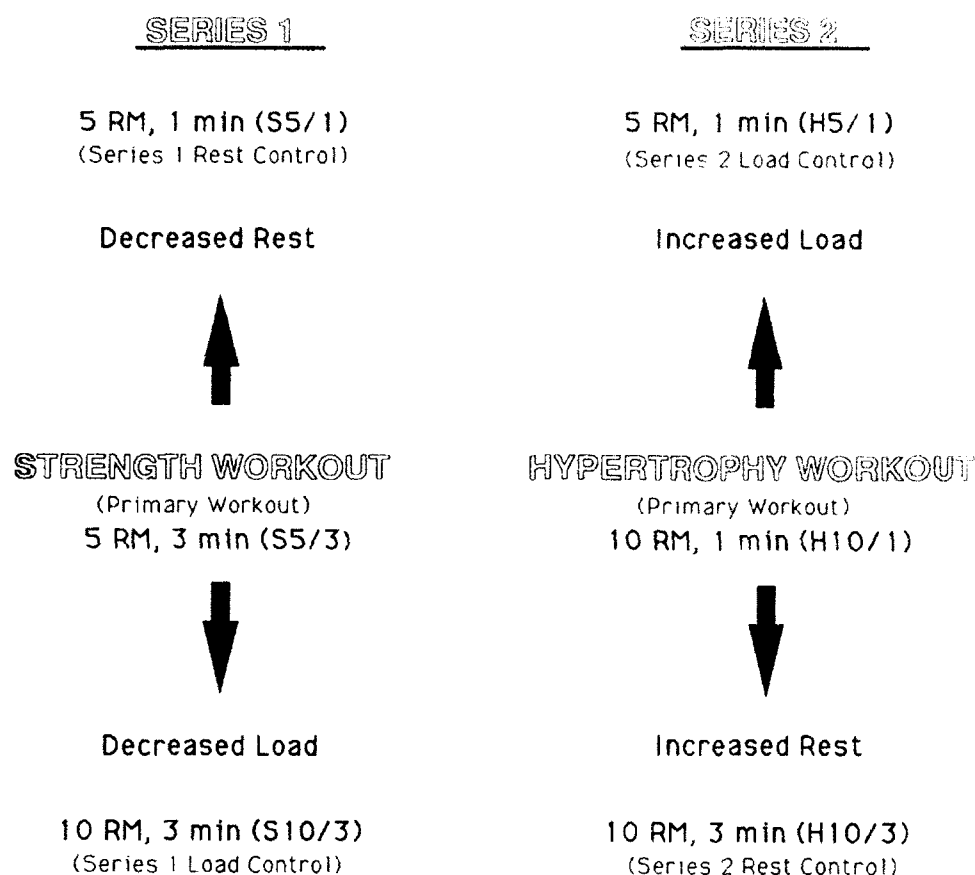


FIG. 1. Schematic diagram of experimental design. RM, repetition maximum.

all protocols. The matching of total work between workouts was performed by a computer program that, given a specific exercise, weight, and number of repetitions, calculated the number of repetitions required to produce the same total work as in the primary protocol in which a different weight was used. Lifting work was calculated as weight \times vertical distance moved per repetition \times number of repetitions. The program took into consideration the vertical distance moved by both the iron plates and the centers of gravity of the lifter's body segments. These distances were obtained from measurements on the subjects and equipment when the subjects were in the starting and ending exercise positions. Anthropometric tables were used to locate body segment centers of gravity

and estimate body segment weights from total body weight (38).

Experimental protocol. One week separated each randomized experimental protocol. Subjects refrained from ingestion of alcohol or caffeine for 24 h and did not perform any strenuous exercise for 48 h before the experimental exercise session. Testing with 1 RM every other week demonstrated that no strength changes occurred over the course of the study. In addition, aerobic exercise was limited to two sessions per week, with no training effects observed over the course of the study.

All venous blood samples were obtained with the subjects in a slightly reclined seated position. Testing was always conducted at the same time of day to reduce the effects of any diurnal variations on hormonal concentrations. Before a resting blood sample was obtained, a 20-min equilibration period was utilized. Subjects knew they would not start exercising until 10 min after the resting blood sample was obtained. This procedure was shown during pilot testing to eliminate any significant anticipatory increases in hormonal responses, sometimes thought to affect the examination of exercise responses. Water intake was allowed ad libitum throughout the exercise protocols and recovery. The venous blood samples were obtained from an indwelling cannula in a superficial arm vein kept patent with isotonic saline (30 ml/h). Blood samples were obtained preexercise, midexercise (i.e., after 4 exercises), immediately postexercise, and at various time points (i.e., 5 min–48 h) after the exercise session, depending on the specific blood variable examined. Whole blood was processed, and where appropriate,

TABLE 1. *Experimental heavy-resistance exercise protocols*

Exercise Order	RM and No. of Sets	
	Series 1 (S5/3)	Series 2 (H10/1)
1) Bench press	5 RM \times 5 sets	10 RM \times 3 sets
2) Double leg extension	5 RM \times 5 sets	10 RM \times 3 sets
3) Military press	5 RM \times 3 sets	10 RM \times 3 sets
4) Bent-leg incline sit-ups	5 RM \times 3 sets	10 RM \times 3 sets
5) Seated rows	5 RM \times 3 sets	10 RM \times 3 sets
6) Lat pulldown	5 RM \times 4 sets	10 RM \times 3 sets
7) Arm curls	5 RM \times 3 sets	10 RM \times 3 sets
8) Leg press	5 RM \times 5 sets	10 RM \times 3 sets

All exercises were performed on a Universal weight machine, except exercises 4 and 7, which utilized free weights. RM, repetition maximum.

serum and plasma samples were stored in an ultralow freezer at -120°C until analyses were performed. Ratings of perceived exertion (RPE), utilizing the Borg CR-10 scale designed to accommodate primarily anaerobic exercise, and heart rate via electrocardiogram were obtained immediately after each exercise set (31).

Biochemical analyses. Whole blood lactate concentrations were determined in duplicate via a lactate analyzer (model 640, Wolverine Medical, Grand Rapids, MI). Hemoglobin was analyzed in triplicate using the cyanmethemoglobin method (Sigma Chemical, St. Louis, MO), and hematocrit was analyzed in triplicate utilizing a standard microcapillary technique. The percent changes in plasma volume were calculated according to equations by Dill and Costill (11). Serum creatine kinase (CK), creatinine, serum urea, and plasma ammonia were all determined in duplicate using colorimetric assay methods and a Gilford Stat Star spectrophotometer (Sigma Chemical). Intra- and interassay variances were all <5 and 8% , respectively. Except for hemoglobin, all serum and plasma samples were run in duplicate and were decoded only after analyses were completed (i.e., blinded analyses). For ammonia and hormonal analyses, blood was collected into prechilled plastic syringes containing EDTA (1.2 mg/ml whole blood), mixed gently, and centrifuged at $1,500\text{ g}$ at 4°C for 15 min. Plasma determinations of the different immunoreactivity values were accomplished with the use of a Beckman 5500 gamma counter and on-line data reduction system. The plasma β -EP radioimmunoassay (RIA) procedure (^{125}I liquid-phase RIA with prior column extraction; INCSTAR, Stillwater, MN) is described in detail elsewhere (24). Cross-reactivity with β -lipotropin was $<5\%$. The interassay variance was 8.1% , and the intra-assay variance was $<4.5\%$. Serum cortisol concentrations were assayed utilizing a solid-phase ^{125}I RIA technique (Diagnostic Products, Los Angeles, CA). The interassay variance was $<6.6\%$, and the intra-assay variance was $<4.1\%$. All hormonal variables were corrected for plasma volume shifts.

Statistical analyses. Statistical evaluation of these data was accomplished utilizing a multivariate analysis of variance with repeated measures. Subsequent post hoc pairwise differences were determined using Tukey tests. Pearson product-moment correlations were utilized to examine various bivariate relationships. Dependent t tests were used for comparisons of the total work of the two series of heavy-resistance exercise protocols. The significance level for this study was $P < 0.05$.

RESULTS

The most prominent finding in this study was that the H10/1 protocol stimulated the highest magnitude of response for most of the variables examined. No significant differences were observed in resting baseline values between exercise protocols for any of the blood variables examined.

β -EP and cortisol. The responses of plasma β -EP to the various heavy-resistance exercise protocols are shown in Fig. 2A. Significant increases in plasma β -EP were observed only for the H10/1 protocol. These elevations were significantly greater than any of the other re-

sponses for the various exercise protocols at these time points, indicating dramatic effects in response to changing the rest period length (i.e., from 1 to 3 min) or increasing the resistance load (i.e., from 10 to 5 RM). No differences were observed in total work between the appropriate S and H series protocols. Furthermore, no differences from preexercise values were observed in resting values 24 and 48 h after the exercise sessions.

The acute responses of serum cortisol (Fig. 2B) followed a pattern similar to that observed for β -EP. The H10/1 protocol produced significant increases, which were greater than all other exercise protocols at the respective times. Similarly, an increase in the rest period length or the resistance load resulted in marked reductions in the serum cortisol concentrations. Again, no differences were observed for appropriate total work comparisons between the S and H series.

Lactate and ammonia. Figure 3 shows the responses of blood lactate (A) and plasma ammonia (B). Significant whole blood lactate increases above resting values were observed at various times during and after exercise in each exercise protocol. The magnitude of increase was again greatest with the H10/1 protocol.

The effects of rest period change in the S series showed that decreasing the rest period to 1 min (from S5/3 to S5/1) significantly increased the lactate concentrations over S5/3 values at midexercise and 0, 5, and 15 min postexercise. In the H series, increasing the rest period from 1 (H10/1) to 3 min (H10/3) significantly decreased lactate values at midexercise and 0, 5, and 15 min postexercise.

In the S series, a significant reduction at midexercise was the only difference observed in whole blood lactate when the load was lightened (from S5/3 to S10/3). Conversely, in the H series, when the load was increased from 10 to 5 RM (from H10/1 to H5/1), significant decreases were observed in lactate at midexercise and 0, 5, and 15 min postexercise. Appropriate total work comparisons (i.e., S5/1 vs. H5/1 and S10/3 vs. H10/3) showed no pairwise differences in lactate between S5/1 and H5/1, but H10/3 values at midexercise and 0, 5, and 15 min postexercise were significantly greater than S10/3 values at those time points.

For plasma ammonia (Fig. 3B), no significant increases were observed above rest for any of the time points examined in the S series protocols (*left panel*). In the H series, significant increases were observed above rest for H10/1 at midexercise and for H10/1 and H5/1 immediately and 5 min postexercise. Again, similar to lactate, the magnitude of increase was greatest with the H10/1 exercise protocol. Total work comparisons (i.e., S5/1 vs. H5/1 and S10/3 vs. H10/3) showed pairwise differences in ammonia, inasmuch as values for H5/1 were greater than those for S5/1 immediately, 5 min, and 24 h postexercise. S10/3 ammonia values 24 and 48 h postexercise were greater than corresponding concentrations for H10/3.

In the S series, decreasing the rest period to 1 min (S5/3 to S5/1) had no effect on plasma ammonia results. In the H series, increasing the rest period from 1 (H10/1) to 3 min (H10/3) significantly decreased ammonia values

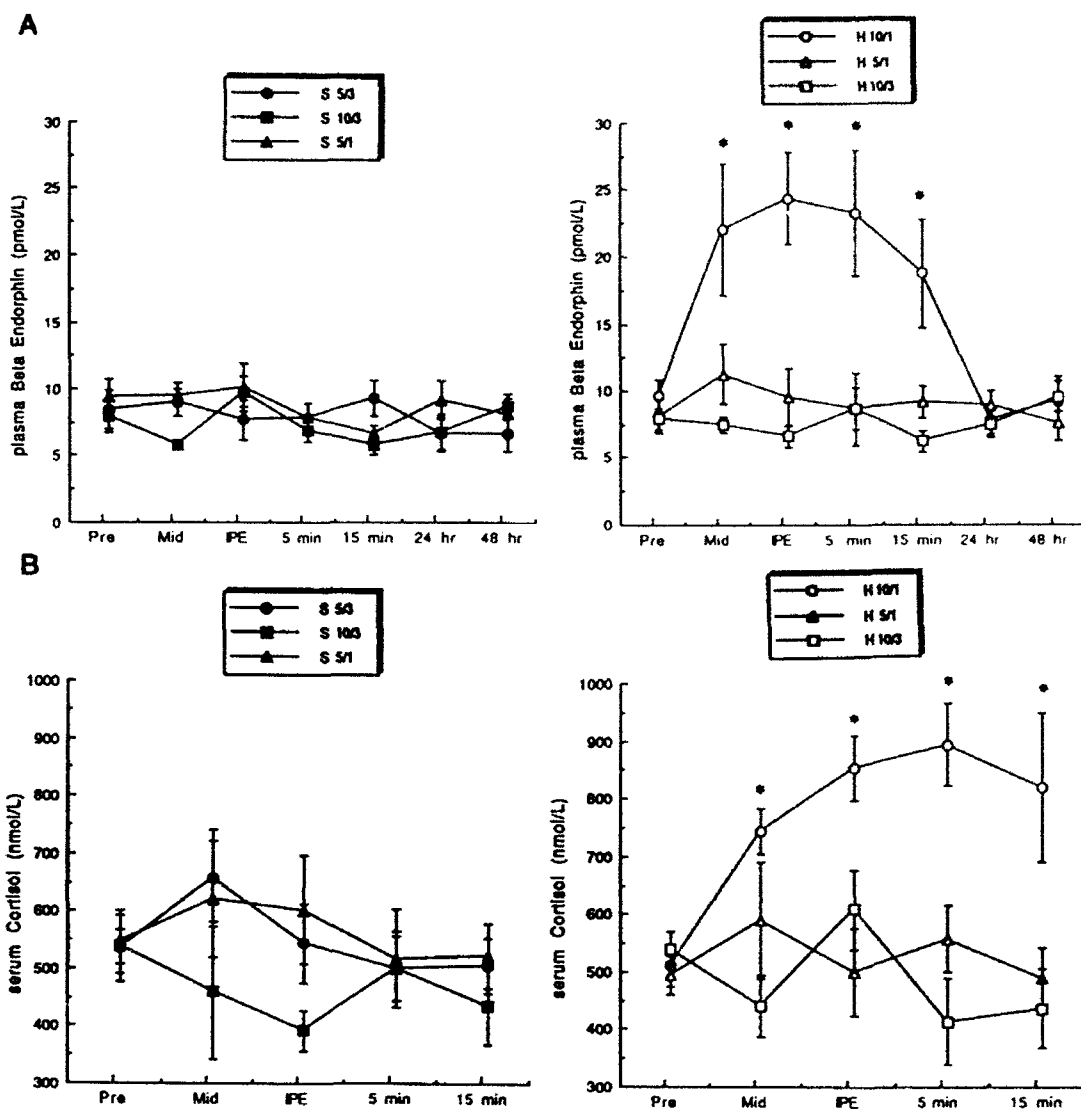


FIG. 2. Plasma β -endorphin (A) and cortisol (B) during strength (S, left) and hypertrophy (H) protocols (right). Values are means \pm SE. Pre, preexercise; Mid, midexercise; IPE, immediately postexercise. * Significant difference ($P < 0.05$) from corresponding preexercise values.

at midexercise and immediately and 5 min postexercise. In the S series, no significant changes were observed when the resistance load was lightened (from S5/3 to S10/3). Conversely, in the H series, when the load was increased (from H10/1 to H5/1), significant decreases were observed in plasma ammonia concentrations at midexercise and immediately and 5 min postexercise.

Creatinine and urea. No significant changes from resting values or differences between any of the heavy-resistance exercise protocols were observed for either serum creatinine (range 200.3 ± 15.91 to $350 \pm 84.86 \mu\text{mol/l}$) or serum urea (range 13.5 ± 7.2 to $19.52 \pm 5.8 \text{ g/dl}$) concentrations measured immediately postexercise and 120 min, 24 h, and 48 h after exercise.

CK. In Fig. 4 the responses of serum CK are presented. In the S series, significant increases above rest were observed after each protocol at 120 min and 24 h, with only the S5/3 and S5/1 values remaining elevated at 48 h. For S5/1, CK concentrations were significantly greater at 120 min and 24 h than for any of the other S protocols. In

the H series, significant increases above rest were observed for H10/1 and H5/1 120 min postexercise. All three protocols increased at 24 h, whereas only the H10/1 and H5/1 values remained elevated at 48 h. At 120 min and 24 h postexercise, the H10/1 protocol resulted in serum CK concentrations significantly greater than for any other heavy-resistance exercise protocol.

Figure 5 shows the response of RPE (A) and heart rate (B) for the different exercise protocols. Except for an increase in RPE for S5/3 immediately after the workout, no further significant changes were observed over the course of each of the exercise protocols. Still, in the S series, RPE for S5/1 was significantly greater than for S5/3 and H10/3 after exercise 1 and midworkout. Immediately after the exercise protocol, RPE for S5/3 and S5/1 were greater than for S10/3. In the H series, RPE for H10/1 and H5/1 were greater at all corresponding time points than for H10/3. For total work comparisons, RPE were greater for H10/3 than for S10/3, whereas no significant differences were observed between S5/1 and H5/1.

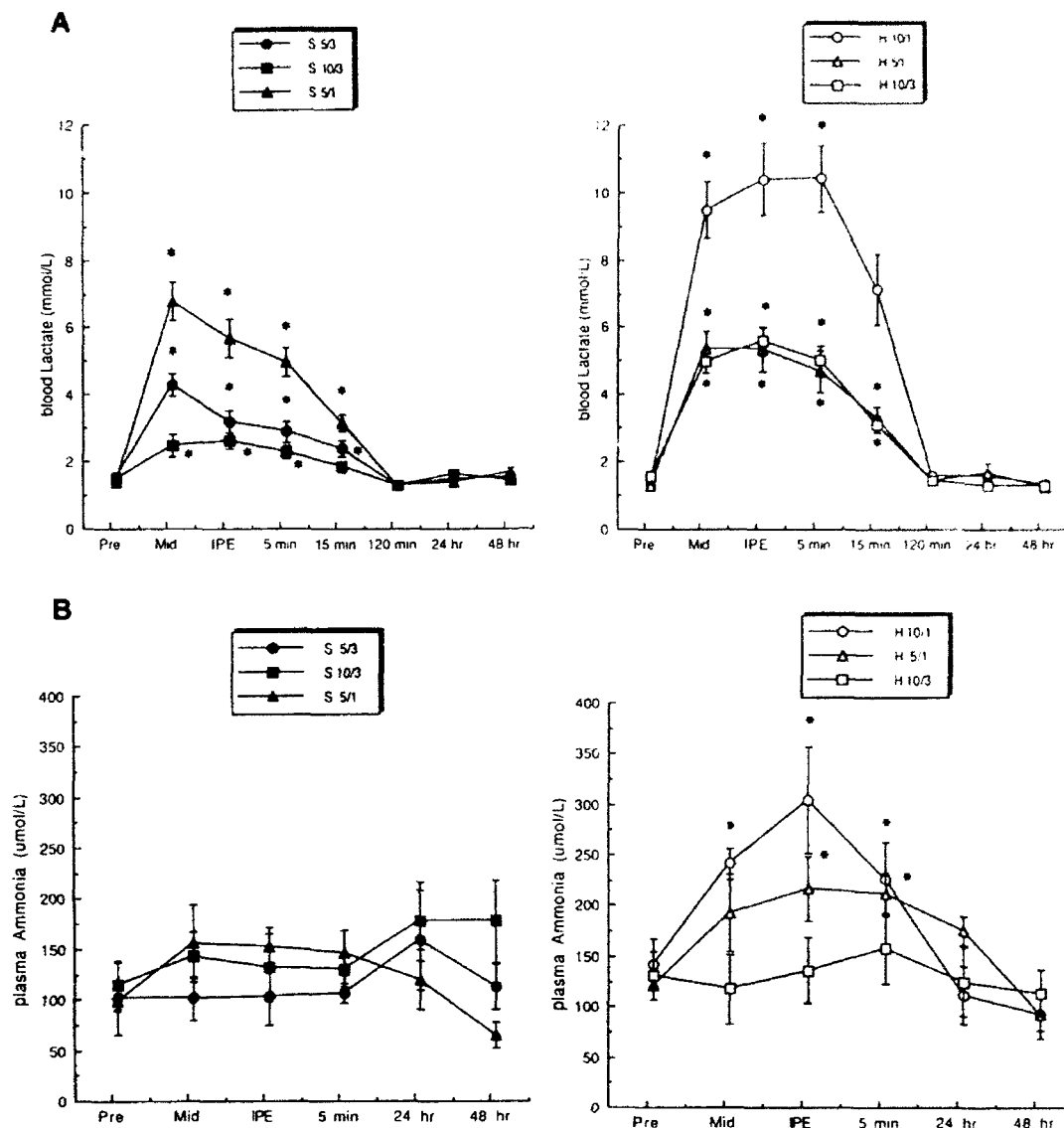


FIG. 3. Whole blood lactate (A) and plasma ammonia (B) during S (left) and H protocols (right). Values are means \pm SE. * Significant difference ($P < 0.05$) from corresponding preexercise values.

The heart rate responses shown in Fig. 5B demonstrated expected increases after exercises 4 and 8. Only for H10/1 did heart rate remain above preexercise values for the entire exercise protocol.

The greatest percent changes in plasma volume were observed pre- to postexercise and were as follows: S5/3, $-6.69 \pm 4.28\%$ (SD); S10/3, $-3.43 \pm 4.15\%$; and S5/1, $-4.87 \pm 2.46\%$; H10/1, $-8.77 \pm 5.42\%$; H5/1, $-3.44 \pm 2.64\%$; and H10/3, $-2.87 \pm 2.41\%$.

Simple linear regression analyses among all the dependent variables were performed for each of the specific individual heavy-resistance exercise protocols. The first statistical approach utilized all the time points in the analyses, and the second approach examined the correlational relationship at each of the specific time points within each exercise protocol. This was done to determine significant correlations within a specific heavy-resistance exercise protocol for the dependent variables measured in this investigation. The following are the only significant correlations observed within each of the

various heavy-resistance exercise protocols. When the relationship between plasma β -EP and whole blood lactate responses was examined using all the time points from just the H10/1 protocol, a significant correlation was observed ($r = 0.82$). Ammonia was correlated to whole blood lactate in the H10/1 protocol ($r = 0.83$). CK concentrations at 24 h were significantly correlated with the highest serum cortisol concentrations measured 5 min postexercise when only the data from the H10/1 protocol were examined ($r = 0.84$).

DISCUSSION

The exact mechanism(s) responsible for exercise-induced increases in β -EP, adrenocorticotrophic hormone (ACTH), and cortisol remain speculative and may differ between submaximal and maximal (i.e., $\dot{V}O_{2\max}$) exercise stressors (18, 22). Furthermore it has been known for a long time that differences exist between the responses of β -EP in the central nervous system and peripheral circu-

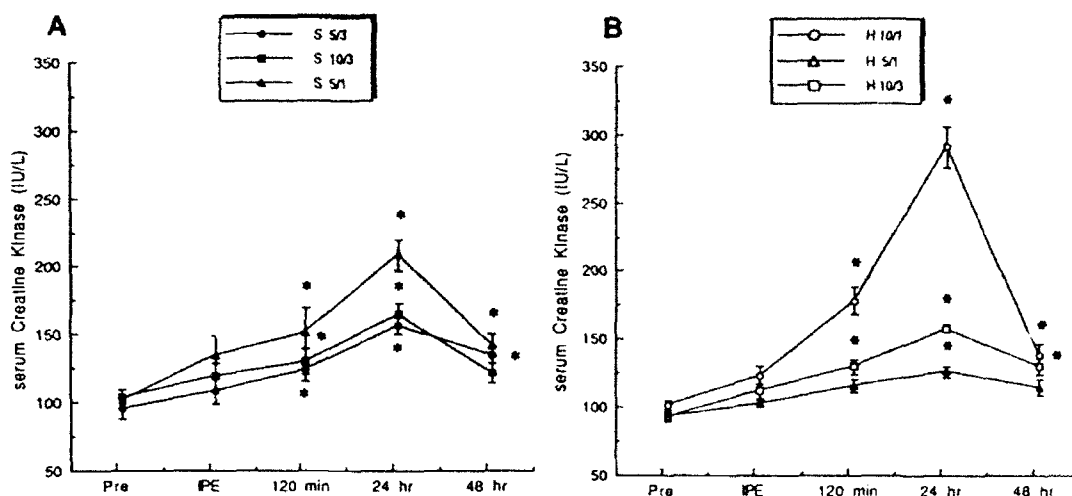


FIG. 4. Serum creatine kinase during S (A) and H protocols (B). Values are means \pm SE. * Significant difference ($P < 0.05$) from corresponding preexercise values.

lation (33). Thus, implications to central mechanisms with exercise stress remain beyond the scope of this investigation, which examined the possible influences on the peripheral β -EP concentrations in the blood. Although short-term anaerobic exercise to exhaustion has been shown to increase plasma β -EP and serum cortisol concentrations in the blood, intensities that are very high and of short duration do not appear to elicit a change in the peripheral plasma concentrations (28). Little is known about responses of plasma β -EP to repetitive and very-high-intensity exercise, as characterized by heavy-resistance exercise.

The primary finding in the present study was that only one of the six heavy-resistance exercise protocols examined resulted in a significant increase in plasma β -EP and serum cortisol concentrations. In general, these data indicate that specific configurations of heavy-resistance exercise programs may differentially influence mechanisms that perturb peripheral plasma concentrations of these hormones. Because exercises used in each heavy-resistance exercise protocol remained identical for each protocol examined, it appears that the exercise stimulus most effective in causing dramatic increases in plasma β -EP is characterized by longer-duration sets (i.e., 10-RM load) and shorter rest periods between sets and exercises. As pointed out by Goldfarb et al. (19), there might be a threshold of exercise intensity above which plasma β -EP concentration is a function of both the duration and intensity of exercise. Our data indicate that, with the type of high-intensity resistance exercise used in this investigation, the number of repetitions that the resistance (i.e., intensity) allows (e.g., 5 or 10 RM) in the set (i.e., duration) and interset rest period length are the primary determinants of physiological stress. The most marked changes in the hormonal responses were produced when the resistance allowed only 10 RM and when short rest periods were utilized. Our experimental design is limited in describing any intensity/duration dual threshold. Further study is needed to establish a continuum of responses of plasma β -EP to heavy-resistance exercise with use of a combination stress consisting of different

set durations (as determined by the RM resistance used) and interset rest periods.

Although the exact mechanisms responsible for exercise-induced increases in plasma β -EP in response to high-intensity exercise remain unknown, it has been suggested by Kjaer et al. (22) that nerve impulses from the motor centers in the brain may directly enhance the activity of higher neuroendocrine centers. In our previous study with essentially the same subject population, growth hormone also responded markedly to the H10/1 protocol (26). Yet, unlike growth hormone responses, plasma β -EP concentrations responded to only one heavy-resistance exercise protocol. Thus a general stress response to heavy-resistance exercise of the hypothalamic-pituitary axis does not appear plausible. In addition, cortisol demonstrated a similar response pattern with β -EP over the first 15 min of recovery, and this response is most likely reflective of ACTH release with high-intensity exercise, which has been shown to typically follow β -EP responses (25, 28). Cortisol has been observed to be responsive to short-rest (i.e., 10–60 s) heavy-resistance exercise protocols (27).

In a recent study by Farrell and co-workers (13) examining a number of different stressors, maximal handgrip exercise produced an exaggerated pressor response when opioid actions were blocked by naloxone, thus suggesting that opioid peptide systems may play a role in modulating sympathetic responses. Although the study by Farrell and co-workers (13) examined much smaller muscle group activity, such data do imply that the sympathetic nervous system responses and pressor activity may be involved with the increases observed in β -EP in response to heavy-resistance exercise. The magnitude of the pressor response with large muscle group heavy-resistance exercise has been shown to involve marked pressor and sympathetic nervous system involvement (17, 22, 23, 27, 30). The combination exercise stress in the H10/1 heavy-resistance exercise protocol of longer-duration (10-RM) sets and shorter rest periods (1 min) allowed less cardiovascular recovery between sets. The use of a 10-RM resistance for each exercise may have maintained a higher

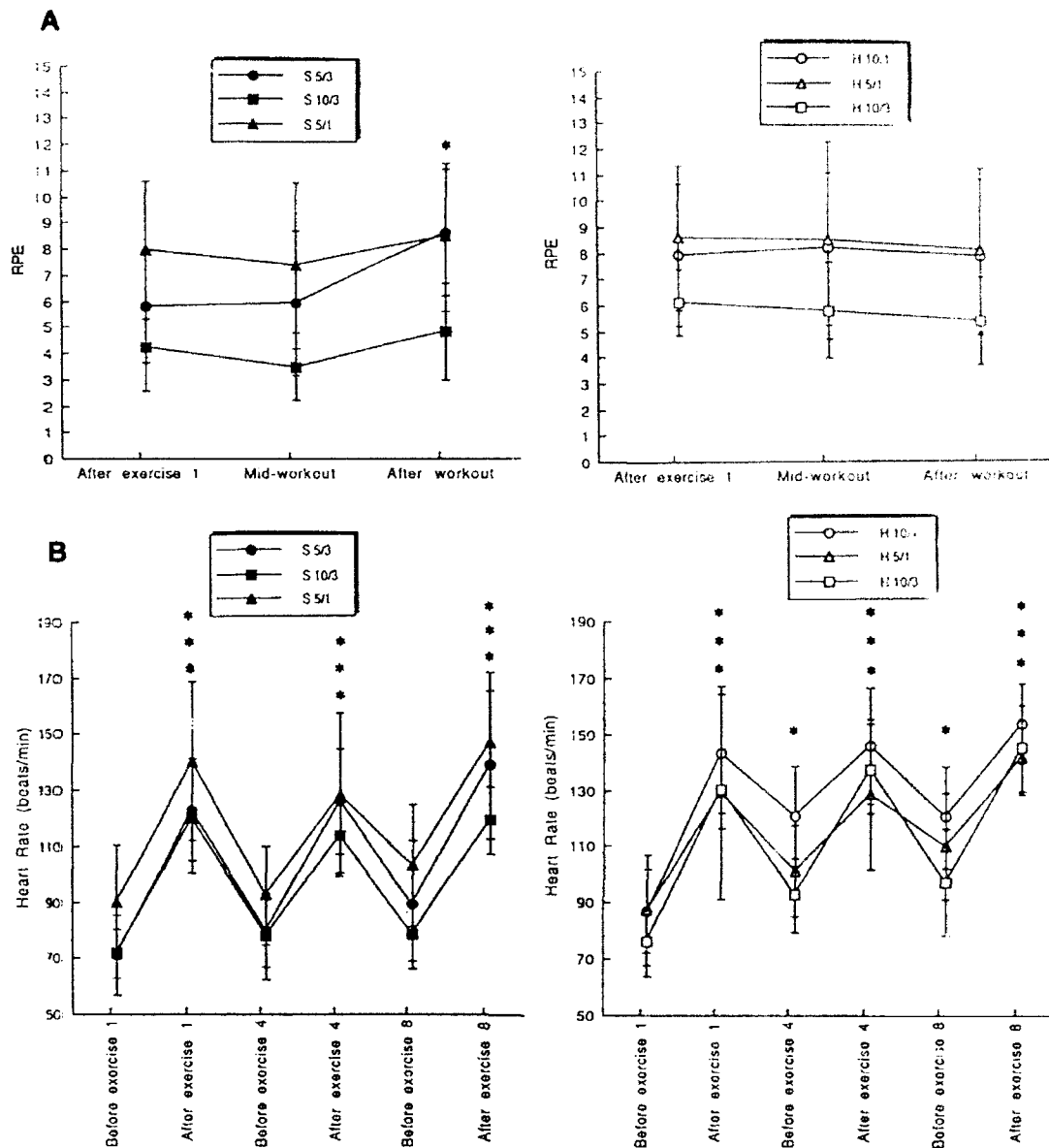


FIG. 5. Ratings of perceived exertion (RPE, A) and heart rate (B) during S (left) and H protocols (right). Values are means \pm SE. * Significant difference ($P < 0.05$) from corresponding preexercise values.

blood pressure response throughout the exercise session. Some evidence to support this speculation may be observed in the heart rate responses of the subjects, which remained significantly elevated above the resting level for the entire H10/1 protocol. The longer duration of each set with resistances of 70 and 80% of the 1 RM have been shown to result in higher arterial blood pressure responses than shorter-duration sets using maximal or near-maximal (90–100% of the 1-RM) resistances (17). It remains to be directly demonstrated whether the higher plasma β -EP concentrations observed in this study for the H10/1 protocol are related to such a modulation and maintenance in the magnitude of sympathetic excitation and pressor responses during the exercise protocol.

The influence of "anaerobic factors" as a systemic stimulus for increases in proopiomelanocortin (POMC) peptides in the plasma has been suggested (14, 25). Despite significant correlational relationships between in-

creases in β -EP and blood lactate, a cause-and-effect relationship has never been demonstrated. In this investigation, a significant correlation ($r = 0.82$) was observed between plasma β -EP concentrations and whole blood lactate only with the H10/1 protocol, which demonstrated the highest blood lactate responses. It is interesting that when the load was increased, the whole blood concentration of lactate was decreased, most likely due to the short duration of exercise associated with the 5-RM set. We did not observe any significant relationships between β -EP and any of the other blood variables such as ammonia, urea, or creatinine. It appears that mechanisms related to acid-base status of the blood are in some way associated with changes in plasma β -EP. Previously, we demonstrated that increases in lactate alone do not result in increased plasma β -EP concentrations (28). Significant correlations with β -EP and lactate have been observed primarily for longer-duration (i.e., 5 min) high-

intensity exercise (25). Conversely, with submaximal single-bout high-intensity exercise of very short duration (<3 min), such bivariate relationships between lactate and β -EP have not been consistently observed (20, 28). Thus, these data, along with the data from previous investigations, start to indicate that the exercise stress may need to dramatically increase and maintain lactate concentrations in the blood in order to challenge the acid-base status and buffering capacity and influence endorphin mechanisms. When appropriately challenged, various physiological mechanisms related to metabolic acidosis may contribute to increases in β -EP. Potential mechanisms may start at the molecular level by increasing the processing of the POMC molecule because of more optimal conditions of a lower pH for converting-enzyme activities involved in the processing of the polypeptide precursor (7).

Although marked increases in β -EP occurred during the H10/1 protocol, resting concentrations of these hormones 24 and 48 h postexercise were not different from preexercise values. Peak cortisol responses 5 min postexercise and peak CK concentrations 24 h postexercise were significantly correlated ($r = 0.84$) for the H10/1 protocol. The magnitude of CK changes was much lower than those observed with human eccentric muscle tissue damage models (1, 8). Still, it is interesting that, with concentric limited resistances used in this investigation, the highest responses were consequent to the H10/1 protocol, which utilized lighter resistances but had the most dramatic increases in blood lactate, β -EP, and cortisol. The catabolic role of cortisol and its relationship to muscle tissue remodeling may account for our present observation of a significant correlation between cortisol and CK (2, 18, 32, 34). In general, many body-building programs, primarily directed toward more dramatic increases in muscle hypertrophy, are similar to the H10/1 protocol (27).

The primary finding of this investigation was that the patterns of response of plasma β -EP and serum cortisol were similar during heavy-resistance exercise. Still, this response was different, depending on the specific type of exercise protocol used. Finally, when heavy-resistance exercise is utilized as an exercise modality, it appears that the durations of the force production (10 RM) and interset rest period are key factors in the configuration of the exercise stimulus that increases plasma β -EP and serum cortisol concentrations.

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The views, opinions, and/or findings contained in this report are those of the author(s) and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation.

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